

Original article

Neuromuscular control of walking with chronic low-back pain

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SUMMARY. The reported association of low-back pain and musculoskeletal disorders contributed to the examination of the lumbar spine and hip extensor activation patterns in back pain sufferers during walking. Seventeen idiopathic low-back pain male subjects and 16 healthy volunteers participated in the study. Hip joint ROMs in the sagittal plane and neuromuscular activities of erector spinae [L3, T12], gluteus maximus and biceps femoris were recorded on one randomly selected body side in each group. Analysis using the Student's *t*-test revealed significant differences for hip joint range of motion, stride time and significantly earlier onsets of the lumbar spine and hip extensors of the back pain sufferers compared with the healthy controls. It is assumed, that low-back disorders are related to changes of the lumbar spine and hip extensor recruitment pattern.

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INTRODUCTION

Low-back pain is one of the most common musculoskeletal problems in modern society. Demonstrated by the high direct and indirect costs, it also causes major economic problems in industrialized nations (Berger-Schmitt et al. 1996; Maniadakis & Gray 2000). Thus, chronic low-back pain problems have been the reason for many clinical investigations.

Although there is disagreement in the literature with regard to the etiology (White & Gordon 1982; Bernard & Kirkaldy-Willis 1987; Nachemson 1992), it seems evident that idiopathic low-back pain is often associated with musculoskeletal disorders and imbalances in lumbar spine and pelvic stabilization muscles (Schneider 1981; Janda 1984; Liebenson 1990; Bourdillon et al. 1994; Norris 1995). It is

suggested that deficiencies in movement patterns and motor regulation play a major role in the development of musculoskeletal dysfunction (Singer 1986; Jull & Janda 1987, Janda 1992; Bittmann & Badtke 1994). Neural dysregulation due to musculoskeletal pain syndromes might contribute to alterations in the recruitment pattern of various synergistic muscles. Alterations in motor control may cause muscles to be activated in an inappropriate manner (i.e. timing, rate of force development), interfering with a subject's ability to automatically perform adequate movement patterns. Janda postulated in 1978 that at least some cases of low-back pain may occur due to deficiencies in the central nervous control of locomotion. It is assumed that especially disturbances of the activation pattern of the hip extensor and pelvic stabilization muscles are a factor in the genesis of low-back disorders. Even impairments in the peripheral parts of the body seem to be complemented by changes in the central nervous system regulation of the muscles in the lumbo-pelvic region (Bullock-Saxton et al. 1994, Bullock-Saxton 1994; Beckmann & Buchanan 1995).

Prone hip extension is an assessment procedure which has been used by various authors (Pierce & Lee 1990; Lieftring et al. 1991; Janda 1992; Lewit 1992; Badtke et al. 1994; Bullock-Saxton et al. 1994; Vogt & Banzer 1997) to evaluate the neuromuscular

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activation pattern (order of muscle contraction) of surrounding hip muscles. Isolated extension of the hip from the neutral position is normally selected because of its functional importance in stance and locomotion. However, prone hip extension is a open kinetic chain non-weight-bearing position performed by concentric muscle contraction, so joint afferent activity and muscle recruitment strategies will be considerably different from those in gait. Thus, it seems questionable if isolated laboratory test conditions will be able to monitor the authentic muscle recruitment pattern around the trunk and pelvis and identify functional adaptations to back pain.

Although a few back pain studies have already analysed muscle activity during gait (Ahern et al. 1986, Arendt-Nielsen et al. 1995, Arendt-Nielsen 1996) no study has clearly described the muscle firing order of the lumbar and hip muscles in walking. Therefore, it still remains unclear how human musculoskeletal pain modulates motor performance in every day tasks. The aim of the current study was to examine changes in the lumbar spine and hip extensor activation patterns in chronic low-back pain patients in a more functional and complex test situation like walking.

METHODS

Seventeen male subjects (Age: 36.3 ± 2.1 year, Height: 174.7 ± 7.3 cm, Weight: 78.8 ± 14.6 kg) with chronic idiopathic low-back pain (CLBP) diagnosed by a physician (Table 1) and 16 age matched healthy males (Age: 33.7 ± 3.1 year, Height: 178.8 ± 5.2 cm, Weight: 77.2 ± 6.4 kg) (Table 2) participated in the study. Due to the small sample size, and to control for confounding variables, such as gender differences, the study concentrated on one gender only. Individuals were recruited from co-operating rehabilitation clinics and university staff. In both groups, measurements were carried out unilaterally on one randomly selected side of the body.

The visual analogue scale (VAS; 0=no pain and 10=most severe pain) (Triano et al. 1993) and Oswestry Disability Questionnaire (Fairbanks et al. 1980) were used for actual pain intensity and disability ratings. Both of these instruments have

previously been tested for reliability and validity (Deyo et al. 1986, Graver et al. 1998). To ensure that the back pain sufferers experienced at least moderate pain intensities at the time of testing, patients with self-reported pain ratings below 3 (VAS) were not included in the study (Arendt-Nielsen et al. 1996).

Hip joint range of motion in the sagittal plane and neuromuscular activities of lumbar and thoracolumbar erector spinae [L3, T12], gluteus maximus and biceps femoris were recorded unilaterally during treadmill walking (HP-Cosmos[®]-Quasarmed, Germany) at 1.25 m/s. Relative hip flexion and extension was recorded by an electronic goniometer (Biovision[®], Germany) with the axis of rotation aligned to the greater trochanter. The goniometer was calibrated in neutral upright standing. Pregelled (Ag/AgCl) surface electrodes (BlueSensor[®]) with an inter-electrode distance of 20 mm were applied longitudinally over the selected muscles referring to international recommendations (Hermens & Freriks 1997). The reference electrode was attached to the subjects' posterior superior iliac spine. The skin of the recording site was prepared according to the International Society of Electrophysiology and Kinesiology (ISEK) standards (Winter et al. 1980) by shaving as required, sanding, and rubbing with gauze, saturated with alcohol. All electrode cables were lightly secured with tape to reduce any possibility of artefacts produced by cable movement. In order to relate the EMG activity to the instant of heel-strike, pressure-sensitive footswitches were secured at both heels. The subjects had time to practice treadmill walking until they reported that they had become accustomed to the walking conditions. After a rest period of at least 15 min the subjects started to walk again until they reported to feel comfortable. Then data over a minimum of 20 strides were collected for each subject using a multi-channel EMG datalogger system (Biovision[®], Germany; input impedance: 10 G Ω , CMRR: 130 db, RTI noise 8 nV/Hz, gain: 2500, filter: 10 Hz low cut-off, 700 Hz high cut-off, amplifier close to the detection site) operating at 1000 Hz per channel. Muscle on/offset was considered to have occurred when 25 consecutive data points of a sliding window exceeded the current mean baseline by three-standard deviations. The three-standard deviation threshold was selected referring to

Table 1. Inclusion and exclusion criteria for 17 male subjects of CLBP group

Inclusion criteria	Exclusion criteria
Age between 25 and 55, full-time employment	LBP lower than buttocks
LBP limited to the lumbar area and buttocks (between T12 and gluteal folds)	Bowel or urinary tract problems
Low-back pain on at least half the days in a single or in multiple episodes within the past 12 months	Neurological deficit or nerve root tension signs
	Leg length discrepancy > 1 cm
	Vascular insufficiency
	Any major surgery of the spine or lower limbs
	Systemic problems (cancer, cardiovascular, endocrine, etc.)

Table 2. Inclusion and exclusion criteria for male control (normal) group ($n = 16$)

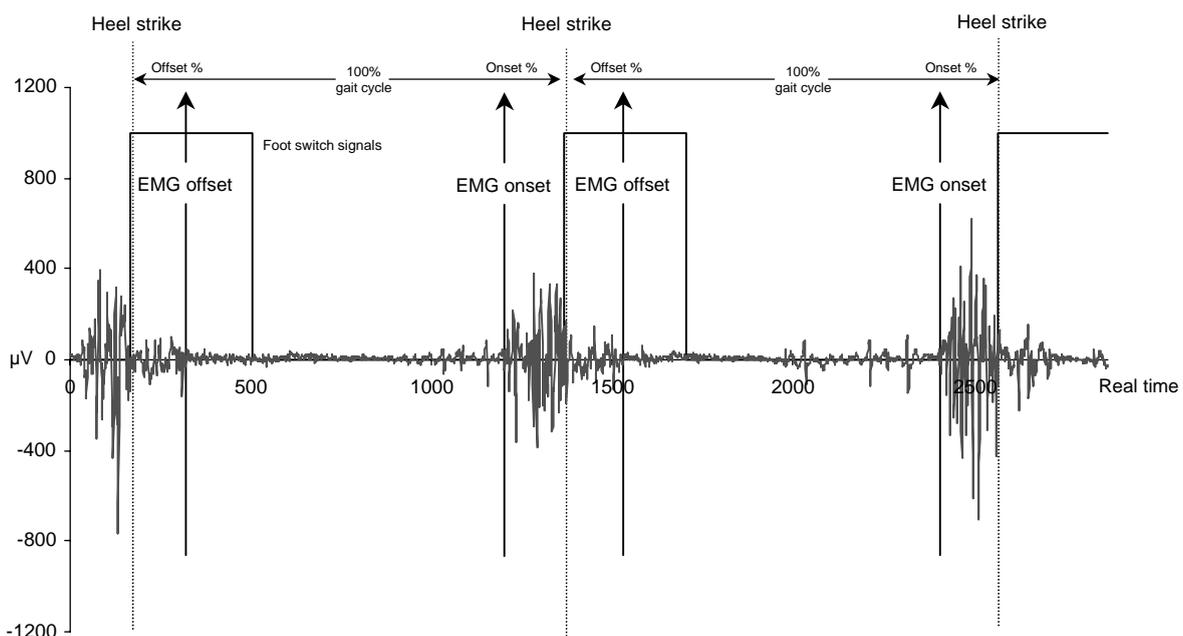
Inclusion criteria	Exclusion criteria
Full-time employment	Previous surgery of the spine or lower extremities
Age between 25 and 55	Any low-back pain in the previous 12 months
Normal spinal curvature and range of motion (total flexion $< 87^\circ$, total extension $< 18^\circ$, lateral flexion $< 24^\circ$, Waddell 1998)	Any loss of time from work for low-back pain
No thoracic or lumbar pathology, including history	Leg length discrepancy > 1 cm
	Any history of arthritis in the lower extremities joints

DiFabio (1987). To account for different cycle durations between subjects selected EMG onset and cessation times were normalized in time for each muscle group and each stride per subject. This was achieved by computing on/offset times relative to the cycle duration of the recording site (heel contact to heel contact corresponding to 0–100%) (Fig. 1). The heel-strike, determined by the footswitch signal ipsilateral to the recording site, was used to define the start/stop of each cycle and to calculate cycle durations. Herewith, the analysis focused on the temporal characteristics (onset and cessation times) of the raw EMG signals during the gait cycle.

To gain additional information from the 'phasic' EMG activity and to concentrate on the shape of the EMG profiles, linear envelopes (full-wave-rectifier followed by a second-order low-pass filter with cut-off at 8 Hz) were calculated (Winter 1984). This reliable method (Kadaba et al. 1989; Kleissen et al. 1997), applied in most gait laboratories (Harris & Wertsch 1994; Whittle 1996), is thought to mathematically model the muscle tension by the use of a single pass second-order low-pass system (Winter

1984, 1990). Due to the time delays introduced by the applied digital filter and the thereby affected occurrence of peaks the current analysis focused on the pattern of the averaged EMG profiles instead of peak characteristics.

The signals were normalized with regard to the stride time (one cycle corresponds to 100%) to maintain timing relative to the walking cycle for the comparison of different subjects. In this way, more than 20 time-normalized EMG profiles formed the data base for the within-subject ensemble average. EMG amplitudes were normalized to the average EMG activity per gait cycle (Yang & Winter 1984). The second averaging procedure resulted in a profile for each group (grand average; CLBP, Controls). Kinematic signals were filtered using a low-pass, zero-lag, critical damped, fourth-order filter (8 Hz cut-off) (Wells & Winter 1980) to allow smoothing without introducing any time delay and time normalized to the ipsilateral heel strike. Student's independent samples *t*-tests were selected to determine significant differences in muscle on/offset, hip movement, and cycle durations between groups.

**Fig. 1**—Normalized EMG on/offset time detection.

Cross-correlation values were computed for pairwise comparisons of the time history of the inter-individual EMG profiles between groups. $P < 0.05$ was regarded as significant.

RESULTS

In chronic low-back pain subjects the self-reported back pain intensity (VAS) ranged from 3 to 5.3 (mean: 3.9) indicating intermediate pain intensities during testing. The subjective disability index (Oswestry-Questionnaire) demonstrated moderate limitations in every day life activities (mean: 26.3%; range: 24–48) within the patient sample. The history of low-back pain ranged from 24 months to 4 years.

Student's t -tests demonstrated significant differences ($P < 0.01$) for hip joint range of motion ($38.3 \pm 9.1^\circ$ vs $25.2 \pm 7.9^\circ$) and stride time (1.06 ± 0.05 s vs 1.03 ± 0.09 s) between healthy controls and back pain patients. Significant EMG onset differences ($P < 0.01$) were found in comparing hip extensors (biceps femoris muscle, gluteus maximus muscle) of the pathological group and the healthy controls. Significant group differences ($P < 0.01$) were also calculated for the onset of both EMG bursts of the bi-phasic activation pattern of the lumbar erector spinae [L3] muscles. Additionally, the analysis revealed a significantly prolonged electrical activity of the gluteus maximus and lumbar erector spinae muscles in the back pain group ($P < 0.01$). No significant group differences in EMG peak characteristics were detected for the thoracolumbar erector spinae [T12]. EMG onset and cessation times of all selected muscles for the independent groups are given in Fig. 2. Cross-correlation values demonstrated almost identical patterns of falling and rising trends

in EMG profiles between groups (Fig. 3). Phase shifts identified by the cross-correlation calculations confirmed the pre-matured EMG activity of the biceps femoris and gluteus maximus in CLBP patients (Fig. 3).

DISCUSSION

The neuromuscular activation of the muscles in the pelvic region plays a primary role for the physiologic coordination and interaction of pelvis, spine and lower limb movements in human gait. Referring to the stabilization concept postulated by Panjabi (1992a,b; Norris 1995), disturbances of the musculoskeletal and fascial system can be prerequisites for or also consequences of pathological syndromes of the spine. Thus, in pain syndromes of the lumbar/sacral/hip region the gluteal and hamstring muscles may play a role which is often overlooked. The recent study, therefore, intends to provide a more detailed look on low-back pain subjects' muscle contractile patterns of lumbar spine and hip extensors in cyclic movements like walking.

Concentrating on patients with a history of back pain, the recent investigation demonstrated reductions in hip flexion/extension movements as well as reduced gait cycle durations. The kinematic changes seen in the current investigation are consistent with those of other authors (Keefe & Hill 1985; Khodadadeh et al. 1988), who also found that back pain patients walked more slowly and took shorter strides. Therefore, the results provide support for the short strided gait and the observations of a more cautious walking pattern in chronic low-back patients (Zebouni et al. 1992; Dananberg 1998).

The detection procedure of relative EMG on/offset times used in the present study tried to account for

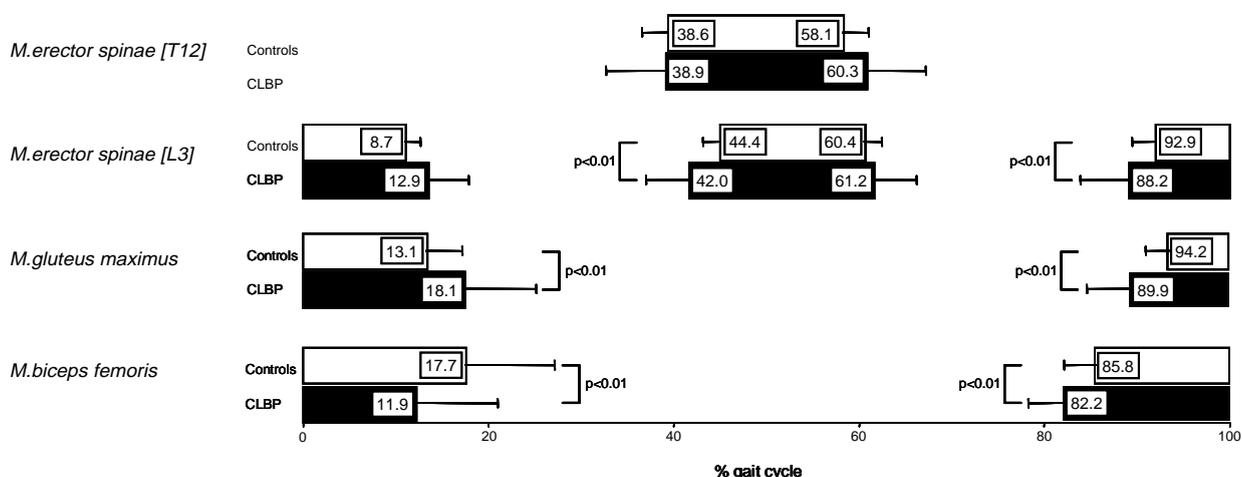


Fig. 2—Average EMG on/offset times of lumbar and thoracolumbar erector spinae [L3, T12], biceps femoris and gluteus maximus for CLBP and healthy controls.

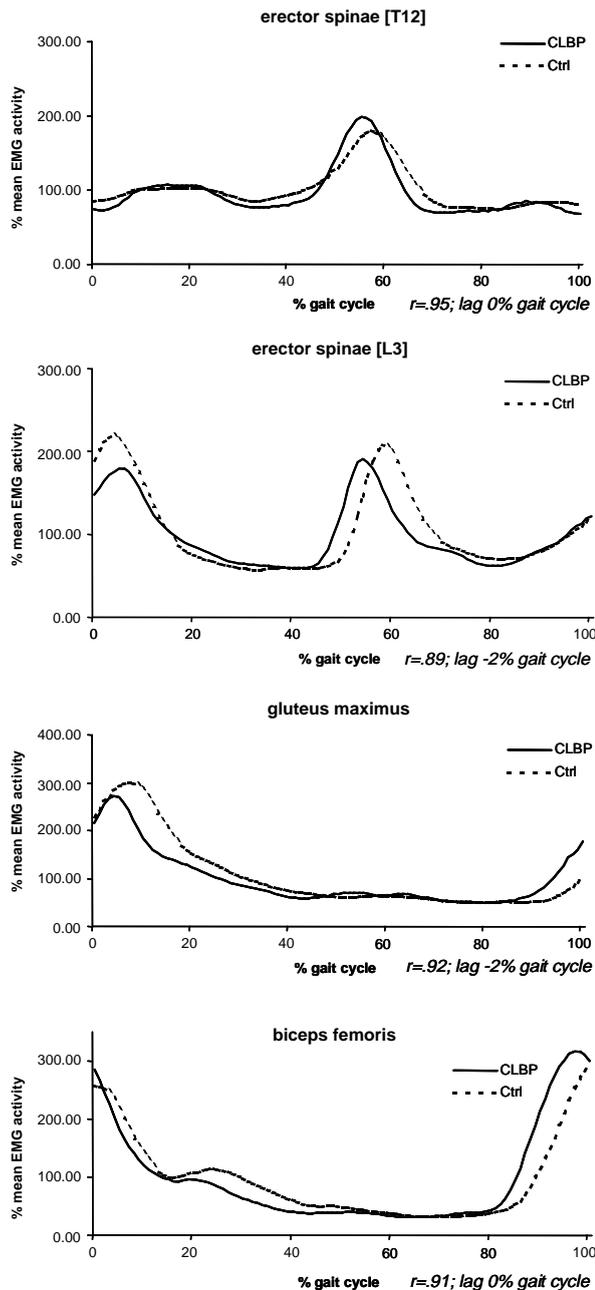


Fig. 3—Average EMG group profiles and cross-correlation values [r] of pairwise comparisons for CLBP patients and healthy controls ($P < 0.001$).

the different stride times between groups and the speed-dependence of the EMG variables. Calculating EMG peak characteristics relative to the individual cycle durations the study revealed significantly pre-matured innervation of the lumbar erector spinae [L3], gluteus maximus, and hamstring muscles in back pain patients compared to the healthy controls. The averaged EMG profiles, produced by linear envelope processing, from the four target muscles are in accordance with findings from other authors (Winter 1984; Kadaba et al. 1989). The cross-correlation values, indicating almost similar patterns

of rises and falls between patients and healthy controls, confirmed the pre-matured activity in lumbar spine and hip extensor muscles in back pain sufferers. Therefore, the results of this study support the idea that some facet of muscle contraction is altered in the presence of low-back dysfunction and do not preclude the idea that back injury is associated with a delayed activation of the gluteus maximus muscle (Janda 1978). Therefore, based on the findings of this study, future work should examine if prone hip extension can be used for the valid assessment of pathological muscle coordination or the evaluation of intervention strategies in the pelvic hip region (Badtke et al. 1994).

In addition to the self-reported pain intensity at the time of testing and the mentioned alterations in the muscle firing pattern, the current findings point towards a protective activation mechanism. Importantly, the pre-matured recruitment strategy of the lumbar spine and hip extensors and the prolonged activity of the gluteus maximus and lumbar spine extensors could be interpreted as a functional adaptation of the neuromuscular system to provide extra stability and to prevent additional pain.

Although, there is no causal evidence that the recent muscle activation patterns are exclusively adaptive factors of idiopathic CLBP, the interpretation of the EMG changes as a functional adaptation to muscle pain are in agreement with results presented by Arendt-Nielsen et al. (1995) and Graven-Nielsen et al. (1997). Their findings of increased lower limb and back muscle activity due to clinical and experimentally provoked muscle pain confirms the hypothesis of pain as a motor output modulator. Herewith, the present results of pre-matured muscle activities would support the 'pain adaptation model' or 'muscle spasm theory' concerning the interaction between motor output and musculoskeletal pain (Collins et al. 1982, Lund et al. 1991). Additionally, the recent concepts of pain and neuronal plasticity point toward long-term adaptations in sense of facilitating changes in pain-related systems (Zieglansberger & Tolle 1993). Therefore, prolonged musculoskeletal pain syndromes might contribute to alterations of dynamic motor stereotypes and motor regulation. Likewise, changes of the neuromuscular coordination in association with functional disturbances and pain itself could be considered as a possible underlying source of recurrent or chronic back pain symptoms. Janda (1978) and also Lewit (1992) consider neuromuscular changes as frequent causes for functional disorders of the spine. In this concept, it has been proposed that musculoskeletal dysfunctions associated with CLBP typically present specific movement patterns of the trunk, pelvis or hips in conjunction with markedly altered activation patterns of the stabilizing muscles of the pelvis, hips, and trunk such as the gluteus

maximus and multifidus (Janda 1984). Changes of the dynamic stereotype of locomotion are attributed to the plasticity of the neuromuscular control system. However, it is unclear whether and how the alterations shown in the present study are associated with 'plastic' changes of the human nervous system. Human locomotion is first controlled segmentally and then becomes progressively more dependent on supraspinal systems (Leonhard 1998). Therefore, it has been postulated that collections of neurons termed central pattern generators (CPG) are responsible for the generation of rhythmic activities, such as bipedal locomotion. It has been suggested that spinal cord CPGs generate the basic locomotor rhythm and supramaximal systems serve to initiate and drive the CPGs (Forssberg 1986). Although no direct evidence exists that the neural substrate for human locomotion changes with pathology, learning and modulation plays a critical role in the attainment and maturation of human bipedal gait. This is reflected in the differences between the gait patterns of human infants and adults as well as in the inter-subject variability of movements. However, this does not provide direct evidence that pathology triggers alterations in the shape of neural connectivity, while changes in the human nervous system can be the result of genetically predetermined neural structures, their connectivity and their physiological functioning as well (Grillner 1985).

Regardless of whether the mentioned changes are consequences or causes of the present pathological situation, out-of-phase activity can cause uneconomic movement coordination and changes of the physiological tissue loading. In such cases, the direction and magnitude of joint forces could be affected and other structures of the locomotor system may become painful. Changed functional conditions due to disturbances or dysfunctions caused by adaptations of the neuromuscular system have been reported frequently in studies of different joint systems (McNair et al. 1992; Davis & Dickhoff-Hoffman 1993; Löfvenberg et al. 1995; Pfeifer & Banzer 1999). The functional importance of a well-coordinated muscular control of the spine and the possibility to influence the intensity and timing of muscle activation has, for example, been shown by Bullock-Saxton et al. (1993).

It can be speculated that subjects with non-specific low-back pain like those investigated in the present study may profit from specific intervention programmes to activate muscles more functionally in different types of motor activities and to achieve adequate muscle balance and coordination. Overlooking the underlying pathology can perhaps contribute to rehabilitation failure and prolong therapeutic interventions. Therefore, future studies have to show, whether it is possible to change the firing patterns of the participating muscles

and which kind of therapy intervention may be adequate.

CONCLUSION

This work was indicated that the analysis of the hip extensor activation pattern in gait may be an essential factor in detecting abnormalities in chronic low-back patients.

Following the presented results, it can be assumed that low-back disorders are related to changes of the hip extensor recruitment pattern. The altered muscle activation pattern could have impact on the physiological loading and alter the direction and magnitude of joint reaction forces. Thus, the pre-matured electrical activity itself could be considered as a possible underlying source of recurrent or chronic back pain symptoms. However, it still remains unclear how human musculoskeletal pain modulates motor performance and it is not known whether abnormalities in muscle function precede the onset of idiopathic low-back pain.

Whether or not muscle disturbances are significant causative factors or late complicating factors of idiopathic LBP, it is concluded that the importance of pelvis stabilization muscles in the etiology of CLBP cannot be denied. More study is needed to obtain information on the behaviour of CLBP patients in different therapy intervention programmes.

References

- Ahern DK, Follick MJ, Council JR, Laser-Wolston N 1986 Reliability of lumbar paravertebral EMG assessment in chronic low-back pain. *Archives of Physical Medicine and Rehabilitation* 67: 762–765
- Arendt-Nielsen L 1996 Quantification of human gait with the presence of low-back pain. In: Hermens HJ, Merletti R, Freriks B (eds) *European Activities on Surface ElectroMyoGraphy. Proceedings of the First General SENIAM Workshop*. Roessingh Research and Development b.v., The Netherlands.
- Arendt-Nielsen L, Graven-Nielsen T, Svorrer H, Svensson P 1995 The influence of low-back pain on muscle activity and coordination during gait: A clinical and experimental study. *Pain* 64: 231–240
- Badtke G, Bittmann F, Sotzko A 1994 Muskelfunktion bei der Hüftextension in der Bauchlage untersucht an Kindern der 2. Klasse. *Man and Medicine* 32: 98–101
- Beckmann SM, Buchanan TS 1995 Ankle inversion injury and hypermobility: Effect on hip and ankle muscle electromyography onset latency. *Archives of Physical Medicine and Rehabilitation* 76: 1138–1143
- Berger-Schmitt R, Kohlmann T, Raspe H 1996 Rückenschmerzen in Ost- und Westdeutschland. *Gesundheitswesen* 58: 519–524
- Bernard TN, Kirkaldy-Willis WH 1987 Recognizing specific characteristics of nonspecific low-back pain. *Clinical Orthopaedics and Related Research* 217: 266–280
- Bittmann F, Badtke G 1994 Bewegungsmuster—primärer Faktor von Fehlentwicklungen des Muskel-Skelett-Systems. *Man and Medicine* 32: 61–65
- Bourdillon JF, Day EA, Bookhout MR 1994 *Spinal Manipulation*, 5th edn. Butterworth-Heinemann, Stoneham, MA, pp. 313–333

- Bullock-Saxton JE, Janda V, Bullock MI 1993 Reflex activation of gluteal muscles in walking. *Spine* 6: 704–708
- Bullock-Saxton JE, Janda V, Bullock MI 1994 The influence of ankle sprain injury on muscle activation during hip extension. *International Journal of Sports Medicine* 15: 330–334
- Bullock-Saxton JE 1994 Local sensation changes and altered hip muscle function following severe ankle sprain. *Physical Therapy* 74(1): 17–31
- Collins GA, Cohen MJ, Naliboff BD, Schandler SL 1982 Comparative analysis of paraspinal and frontalis EMG, heart rate and skin conductance in chronic low-back pain patients and normals to various postures and stress. *Scandinavian Journal of Rehabilitation Medicine* 14: 39–46
- Dananberg HJ 1998. Gait mechanics and their relationship to lower back pain: An outcome study. Third Interdisciplinary World Congress on Low-back and Pelvic Pain. Vienna, November, pp 239–241
- Davies GJ, Dickhoff-Hoffman S. 1993 Neuromuscular testing and rehabilitation of the shoulder complex. *J Sports Phys Ther* 2: 449–458
- Deyo RA, Battie M, Beurokes A 1986 Outcome measures for low-back pain research: A proposal for standardized use. *Spine* 23: 2003–2013
- DiFabio R 1987 Reliability of computerized surface electromyography for determining the onset of muscle activity. *Physical Therapy* 67: 43–48
- Fairbanks JCT, Couper J, Davies JB, O'Brien JP 1980 The Oswestry low-back pain disability questionnaire. *Physiotherapy* 66: 271–273
- Forsberg H 1986 Development and integration of human locomotor functions. In: Goldberger ME, Gorio A, Murray M (eds) *Development and Plasticity of the Mammalian Spinal Cord*. Liviana Press, Padova, pp 53–63
- Graven-Nielsen T, Svensson P, Arendt-Nielsen L 1997 Effects of experimental muscle pain on muscle activity and coordination during static and dynamic motor function. *Electroencephalography and Clinical Neurophysiology* 105: 156–164
- Graver V, Loeb M, Rasmussen F, Lie H, Ljunggren AE 1998 Clinical overall score: Outcome evaluation after lumbar disc surgery, assessments of reliability and validity. *Scandinavian Journal of Rehabilitation Medicine* 30: 227–234
- Grillner S 1985 Neurobiological bases of rhythmic motor acts in vertebrates. *Science* 228: 143–149
- Harris GF, Wertsch JJ 1994 Procedures for gait analysis. *Archives of Physical Medicine and Rehabilitation* 75: 216–225
- Hermens HJ, Freriks B 1997 The state of the art on sensors and sensor placement procedures for surface electromyography: A proposal for sensor placement procedures. SENIAM-Deliverable 5, Roessingh Research and Development b.v, The Netherlands.
- Janda V 1978 Muscles, central nervous motor regulation and back problems. Neurobiologic mechanisms. In: Korr M (ed.) *Manipulative Therapy*. Plenum Press, New York, pp 27–41
- Janda V 1984 Gestörte Bewegungsabläufe und Rückenschmerzen. *Man and Medicine* 22: 74–78
- Janda V 1992 Treatment of chronic pain. *Journal of Man and Medicine* 6: 166–168
- Jull GA, Janda V 1987 Muscles and motor control in low back pain: Assessment and management. In: Twomey LT, Taylor JR (eds) *Physical Therapy of the Low Back*. Churchill Livingstone, New York, pp 253–278
- Kadaba MP, Ramakrishnan HK, Wootten ME, Gainey J, Gorton G, Cochran GVB 1989 Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait. *Journal of Orthopaedic Research* 7(6): 849–860
- Keefe FJ, Hill RW 1985 An objective approach to quantifying pain behaviour and gait patterns in low back pain patients. *Pain* 21: 153–161
- Khodadadeh S, Eisenstein S, Summers B, Patrick J 1988 Gait asymmetry in patients with low back pain. *Neuro-Orthopedics* 6: 24–27
- Kleissen RFM, Litjens MCA, Baten CTM, Harlaar J, Hof AL, Zilvold G 1997 Consistency of surface EMG patterns obtained during gait from three laboratories using standardised measurement technique. *Gait Posture* 6: 200–209
- Leonhard CT 1998 Neural control of human locomotion. In: Leonhard CT (ed.) *The Neuroscience of Human Movement*. Mosby, St Louis, pp 146–175
- Lewit K 1992 *Manuelle Medizin*, 6th edn. Ambrosius, Heidelberg
- Liebenson C 1990 Active muscular relaxation techniques. Part II: Clinical application. *Journal of Manipulative and Physiological Therapy* 13(1): 2–6
- Lieftring V, Hinz K, Seidel W, Conradi E 1991 Objektivierung der Muskelaktivität bei krankengymnastischen Bewegungsabläufen mit Mehrkanalelektromyographie. *Z Phys Rehab Kur Med* 1: 33–37
- Löfvenberg R, Kärrholm J, Sundelin G, Ahlgren O 1995 Prolonged Reaction Time in Patients with Chronic Lateral Instability of the Ankle. *American Journal of Sports Medicine* 4: 414–417
- Lund JP, Donga R, Widmer CG, Stohler CS 1991 The pain-adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity. *Canadian Journal of Physiology and Pharmacol* 69: 683–694
- Maniadakis N, Gray A 2000 The economic burden of back pain in the UK. *Pain* 84: 95–103
- McNair PJ, Wood GA, Marshall RN 1992 Stiffness of the hamstrings muscles and its relationship to function in anterior cruciate ligament deficient individuals. *Clinical Biomechanics* 131–137
- Nachemson AL 1992 Newest knowledge of low-back pain. A critical look. *Clinical Orthopaedics and Related Research* 279: 8–20
- Norris CM 1995 Spinal stabilisation 1. Active lumbar stabilisation—concepts. *Physiotherapy* 81(2): 61–64
- Norris CM 1995 Spinal stabilisation 4. Muscle imbalance and the low back. *Physiotherapy* 81(3): 127–138
- Panjabi MM 1992a The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders* 4: 383–389
- Panjabi MM 1992b The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *Journal of Spinal Disorders* 4: 390–397
- Pfeifer K, Banzer W 1999 Motor performance in different dynamic tests in knee rehabilitation. *Scandinavian Journal of Medicine and Science of Sports* 1: 11–17
- Pierce MN, Lee WA. 1990 Muscle firing order during active prone hip extension. *Journal of Orthopaedics, Sports and Physical Therapy* 12: 2–9
- Schneider W 1981 *Manuelle Therapie degenerativer Erkrankungen der Wirbelsäule*. *Therapeutische Umschau* 38(7): 656–659
- Singer K 1986 Suggestions from the clinic: A new musculoskeletal assessment in a student population. *Journal of Sports and Physical Therapy* 8: 34–41
- Triano JJ, McGregor M, Craimer GD, Deborah LE 1993 A comparison of outcome measures for use with back pain patients; results of a feasibility study. *Journal of Manipulative and Physiological Therapy* 16: 67–73
- Vogt L, Banzer W 1997 Dynamic testing of the motorial stereotype in prone hip extension from neutral position. *Clinical Biomechanics* 12(2): 122–127
- Waddell G 1998 *The Back Pain Revolution*. Churchill Livingstone, London
- Wells RP, Winter DA 1980 Assessment of signal and noise in the kinematics of normal, pathological and sporting gaits. *Proceedings of the Special Conference of the CSB, Human Locomotion I*, London, Ontario, Canada
- White AA, Gordon SL 1982 Synopsis: Workshop on idiopathic low-back pain. *Spine* 7(2): 141–149
- Whittle MW 1996 Clinical gait analysis: A review. *Human Movement Science* 15: 369–387
- Winter DA, Rau G, Kadefors R 1980 Units, terms and standards in the reporting of EMG Research. Report by the Ad Hoc Committee of the International Society of Electrophysiological Kinesiology. Department of Medical Research, Rehabilitation Institute of Montreal, Montreal
- Winter DA 1984 Pathologic gait diagnosis with computer-averaged electromyographic profiles. *Archives of Physical Medicine and Rehabilitation* 65: 393–398
- Winter DA 1990 *Biomechanics and Motor Control of Human Movement*, 2nd edn. Wiley, Ontario, Canada

Yang JF, Winter DA 1984 Electromyographic amplitude normalization methods: Improving their sensitivity as diagnostic tools in gait analysis. *Archives of Physical Medicine and Rehabilitation* 65: 517–521

Zebouni L, Helliwell PS, Howe A, Wright V 1992 Gait analysis in ankylosing spondylitis. *Annals of Rheumatic Disease* 51: 898–899

Zieglgansberger W, Tolle TR 1993 The pharmacology of pain signalling. *Current Opinion in Neurobiology* 3: 611–618